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Twelve-lead electrocardiographic localization of idiopathic premature ventricular contraction origins

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Abstract

The major sites of origins of idiopathic ventricular arrhythmias have been elucidated. Idiopathic ventricular arrhythmias most often present as premature ventricular contractions (PVCs) with a focal mechanism, and commonly occur without structural heart disease. Idiopathic ventricular arrhythmias usually originate from specific anatomical structures, commonly endocardial but sometimes epicardial and exhibit characteristic electrocardiograms (ECGs) based on their anatomical background. There are general and specific ECG characteristics that can localize the site of idiopathic PVC origins. The general ECG characteristics include the bundle branch block pattern, axis, QRS polarity in lead V6, QRS duration, precordial transition, maximal deflection index, and so forth. They can roughly localize the site of idiopathic PVC origins. Several major sites of idiopathic PVC origins are located close to each other, and specific ECG characteristics are helpful for localizing the site of origins more accurately in those PVCs. Twelve-lead surface ECG algorithms usually can localize the site of idiopathic PVC origins with a high accuracy, but their accuracy can be limited by the patients' physique, heart rotation, specific conduction properties, presence of structural heart disease, and so forth. This review describes an overview of the approaches to the 12-lead surface ECG localization of idiopathic PVCs, and also discusses their caveats and limitations.

KEYWORDS

electrocardiogram, idiopathic, localization, origin, premature ventricular contraction

1 | INTRODUCTION

In the past 2 decades, advances in procedural techniques, and an improved understanding of electrophysiological mechanisms and cardiac anatomy, have allowed to reveal the major sites of origins of idiopathic ventricular arrhythmias (Figure 1). Idiopathic ventricular arrhythmias most often present as premature ventricular contractions (PVCs). PVCs present with a focal mechanism, and commonly occur without structural heart disease.[1,2] In such a setting, the activation from a PVC origin propagates normally through the healthy myocardium, and exhibits characteristic electrocardiograms (ECGs) that are specific to the site of the PVC origin.[2,3] Therefore, 12-lead surface ECGs can localize the site of PVC origins with a high accuracy.

In the treatment of PVCs, it is important to localize the site of the PVC origin for several reasons. First, medical treatment of PVCs may be specialized to the sites of the PVC origins with different mechanisms.[4,5] Second, when catheter ablation is indicated, the techniques and equipment used for mapping and catheter ablation of PVCs may differ among the sites of PVC origins.[2,3] Therefore, a preprocedural planning of the mapping and catheter ablation approach based on the ECG localization is important to save procedural time and to reduce the cost and complications.

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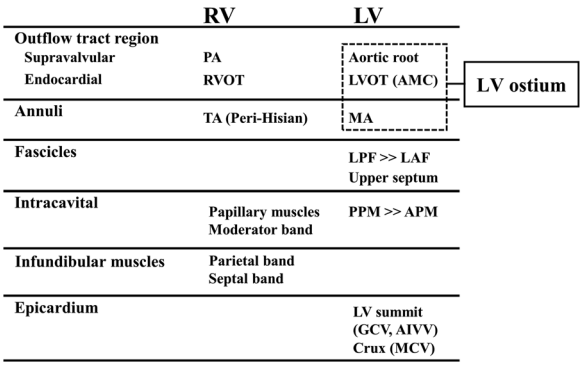


FIGURE 1 Major sites of idiopathic ventricular arrhythmia origins. AIVV, anterior interventricular vein; AMC, aortomitral continuity; APM, anterolateral papillary muscle; GCV, great cardiac vein; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricle; LVOT, LV outflow tract; MA, mitral annulus; MCV, middle cardiac vein; PA, pulmonary artery; PPM, posteromedial papillary muscle; RV, right ventricle; RVOT, RV outflow tract; TA, tricuspid annulus. This figure was modified from reference 13 with permission

This review describes an overview of the approaches to the 12-lead surface ECG localization of idiopathic PVCs, and also discusses their caveats and limitations.

2 | GENERAL CONSIDERATION

In general, the first clue in 12-lead surface ECGs for predicting a site of a PVC origin is a bundle branch block pattern in lead V1.[3] A right bundle branch block (RBBB) pattern clearly suggests an origin in the left ventricle (LV), whereas a left bundle branch block (LBBB) pattern suggests an origin in the right ventricle (RV) or interventricular septum. Second, an inferior axis (dominant R waves in leads II, III, and aVF) suggests an origin in the superior aspect of the ventricle, whereas a superior axis suggests an origin in the inferior aspect. Third, a QS pattern in lead V6 that is located near the apex suggests a PVC origin near the apex because the activation from the PVC origin propagates away from the apex. On the other hand, activation from a PVC origin in the base propagates toward the apex, and reflects an R wave in lead V6. Therefore, an R/S wave amplitude ratio more than 1 in lead V6, suggests an origin in the base (ventricular outflow tract or annuli), whereas an R/S wave amplitude ratio less than 1 suggests an origin in the middle of the ventricle (papillary muscles or left fascicles; Figure 2).[3,6] Fourth, a precordial transition suggests a likely location of a PVC origin. In PVCs with an RBBB pattern, a precordial transition from positive to negative occurs earlier as the site of the PVC origin shifts from the LV base to the apex. In PVCs with an LBBB pattern, a precordial transition from negative to positive occurs earlier as the site of the PVC origin shifts from the RV free wall towards the interventricular septum. Fifth, a narrow QRS complex and absence of a middle or late notching in the QRS complex suggest an origin in the interventricular septum whereas a wide QRS complex and the presence of a middle or late notching in the QRS complex suggest an origin in the ventricular free wall.[3,7] This is because activation from a PVC origin in the interventricular septum excites both ventricles nearly simultaneously, while that from the ventricular free wall activates each ventricle sequentially. A negative QRS polarity in lead I also suggests an origin in the LV free wall.[3,8,9]

Twelve-lead ECGs are very helpful for determining the likely epicardial PVC origins (Figure 3). Because in human hearts, the Purkinje network is located only in the subendocardium, ventricular activation from the epicardial origin requires more time to reach the Purkinje network, resulting in a slow onset of the QRS during epicardial PVCs. Based on this mechanism, several parameters predicting epicardial PVC origins have been proposed; a "pseudo-delta" wave duration >34 ms, QRS duration >200 ms, delayed intrinsicoid deflection of >85 ms, RS complex duration >121 ms, and maximum deflection index (MDI; calculated by dividing the shortest time from the QRS onset to the maximum deflection in any of the precordial leads by the total QRS duration) of >0.54 (Figure 3A).[10,11] When the ventricular activation propagates from an epicardial origin on the LV free wall or ventricular posterior wall, the total activation vector should go from a lateral toward medial or from an inferior toward superior direction, resulting in a QS pattern in lead I or lead aVF (Figure 3B).[12] On the other hand, when ventricular activation propagates from an endocardial origin on the LV free wall or ventricular posterior wall, a part of the activation vector should go toward the lateral or inferior direction, which reflects the activation conducting through the wall of the ventricular muscle toward the epicardium, resulting in the presence of an initial R wave in lead I or lead aVF (Figure 3B). Therefore, a QS pattern in lead I or aVF suggests an epicardial origin in the LV free wall[12,13] or the ventricular posterior wall, respectively (Figure 3B).

All these ECG algorithms are more accurate without structural heart disease than with it because without structural heart disease the ventricular activation propagates away from the PVC origins through healthy ventricular myocardium in a predictable manner.

3 | SPECIFIC CONSIDERATION

Idiopathic PVCs usually originate from specific anatomical structures, and exhibit characteristic ECGs based on their anatomical background (Table 1). If those ECG characteristics are understood, idiopathic PVC origins can be localized with a high accuracy.

3.1 | Ventricular outflow tract origins

The most common site of idiopathic PVC origins is the ventricular outflow tract.[1-3,14,15] The biggest concern about the ECG characteristics of PVCs originating from this region should be how to differentiate whether PVCs originate from the RV outflow tract (RVOT) or LV outflow tract (LVOT) including the aortic sinus of Valsalva (ASV), aortomitral continuity (AMC), and LV summit.[2,3,14-27] The ECG characteristics of idiopathic PVCs originating from the RVOT and LVOT are similar because anatomically, the RVOT and LVOT are located next to each other (Figure 4A). In addition, the

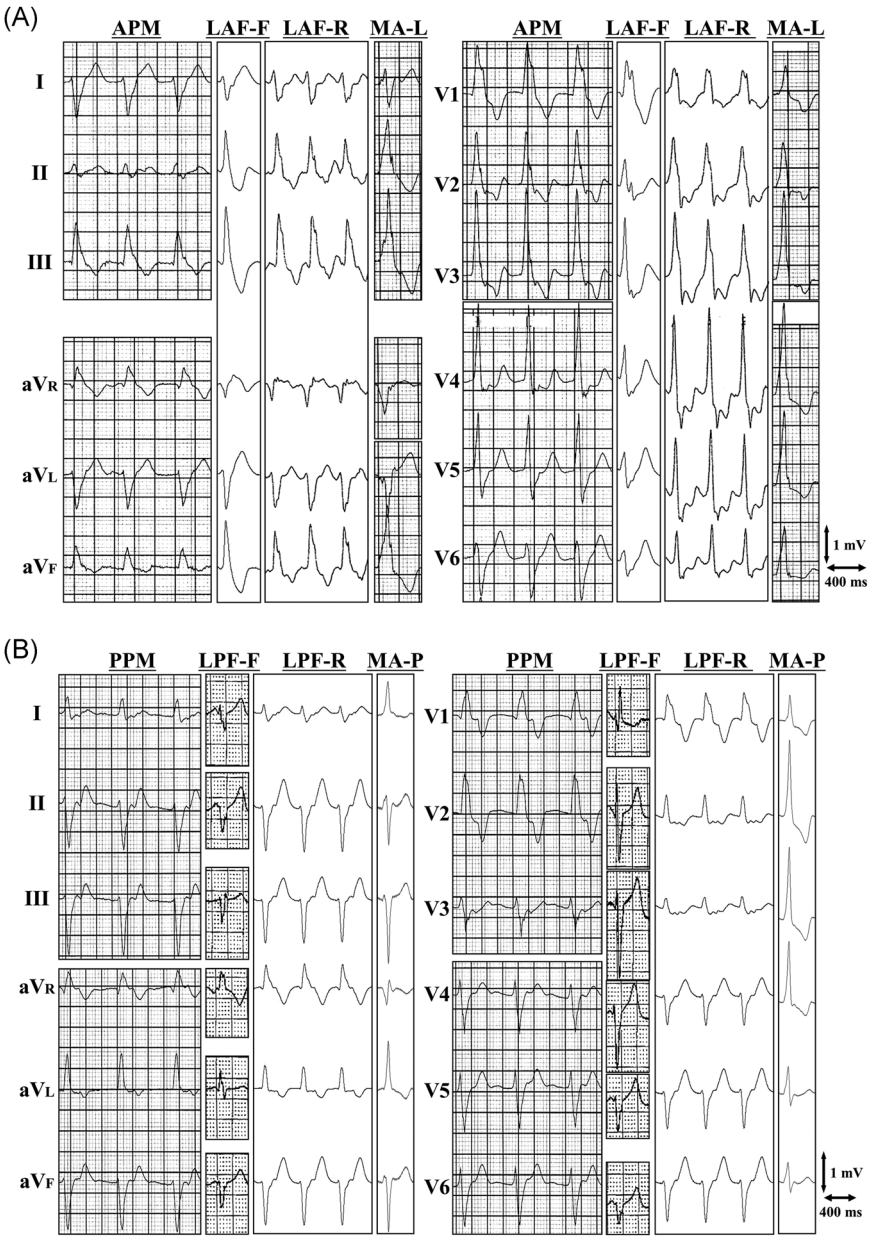


FIGURE 2 The representative 12-lead electrocardiograms of the ventricular arrhythmias originating from the anterolateral (A) and postero-septal (B) regions in the LV. APM, anterolateral papillary muscle; L, lateral portion; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricle; MA, mitral annulus; P, posterior portion; PPM, posteromedial papillary muscle; X-F, R, VAs with a focal or macro-reentrant mechanism. This figure was cited from reference 6 with permission

complex anatomy of these regions may limit the reliability of the ECG algorithms. The ECGs of idiopathic outflow tract PVCs are characterized by positive R waves in all inferior leads and deep S waves in both leads aVR and aVL (almost a QS pattern; Figure 4A). The absence of S waves in lead I is helpful for differentiating RVOT PVC origins from LVOT PVC origins.[17] An RBBB QRS morphology clearly suggests a VA origin on the left side. However, when PVCs present an LBBB QRS morphology, it is often difficult to differentiate RVOT PVCs from LVOT PVCs. Because anatomically, the LVOT is located posterior to the RVOT (Figure 4A), LVOT PVCs exhibit taller and wider R waves in leads V1 and V2 than RVOT PVCs.[13,16] Therefore, the precordial transition is helpful for differentiating RVOT PVCs from LVOT PVCs. When the precordial transition is at lead V4 or later, the PVCs are very likely to originate from the RVOT, and when the precordial transition is at lead V2 or earlier, the PVCs are very likely to originate from the LVOT. However, when the precordial transition is in lead V3, it is most difficult to differentiate RVOT PVCs from LVOT PVCs.[3,21,22,24,26] Multiple ECG algorithms to differentiate RVOT PVCs from LVOT PVCs have been proposed and validated with a high accuracy (Table 2).[17,21,22,24,26] When the accuracy of this kind of ECG algorithms are discussed, there are several concerns to be considered. First, the accuracy of these kinds of ECG algorithms can fluctuate with the patient bias. As mentioned above, any ECG algorithm should be less accurate when the precordial transition is in lead V3 than when it is very early or late. Therefore, the fewer the PVCs that present with a precordial transition in lead V3, the more accurate the ECG algorithm becomes. The readers have to pay attention to the proportion of the PVCs with a precordial transition in lead V3 in the study subjects and the accuracy of the ECG algorithm in only selected PVCs with a precordial transition in lead V3. For PVCs with a precordial transition in lead V3, the specific ECG algorithm focused on those PVCs[21] may

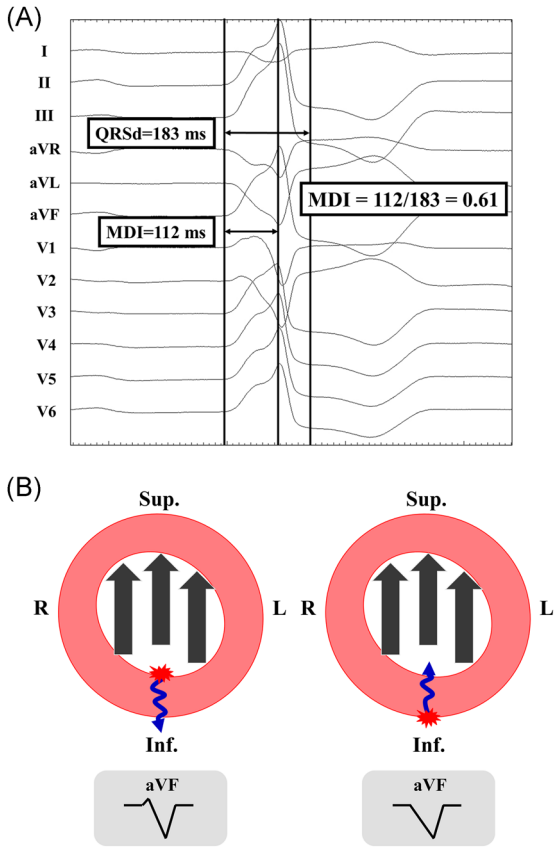


FIGURE 3 A, Twelve-lead electrocardiograms exhibiting a premature ventricular contraction originating from the LV summit and the measurement of the maximal deflection index (MDI). This figure was cited from reference 3 with permission. B, Schema showing the mechanism to explain the difference in the QRS morphology in lead aVF during ventricular arrhythmias with endocardial (left) and epicardial (right) foci. Inf, inferior; L, left; LV, left ventricle; R, right; Sup, superior. This figure was cited from reference 12 with permission

be used together with the standard ECG algorithms for confirmation. Second, these kinds of ECG algorithms should be simple as well as accurate. Complicated ECG algorithms that require multiple measurements and calculations during both sinus rhythm and PVCs may not be recommended. Those ECG algorithms cannot be used for sustained ventricular tachycardia. However, it should be noted that ECG algorithms using measurements during sinus rhythm may be able to adjust for an individual bias such as the physique, heart rotation, and so forth.[21,22] Third, ECG algorithms that use absolute numbers in the ECG measurements as a cut off value may not be recommended because those numbers are susceptible to the patient's physique, sex, race, and so forth. When all these things are taken into consideration, currently, two ECG algorithms may be recommended, the magnitude and width of the R wave or QRS complex in leads V1 and V2 (R/S wave amplitude and duration indexes)[16] (Figure 5A) and the V2S/V3R amplitude ratio[26] (Figure 5B), because they can simply and accurately make a diagnosis by an ECG of a PVC only. The R/S wave amplitude in leads V1 and V2 is measured as the amplitude of the QRS complex peak or nadir to the isoelectric line. The R/S wave amplitude index, calculated from the percentage of the R/S-wave amplitude ratio in lead V1 or V2 (whichever is greater), is considered more useful than the R/S wave amplitude ratio alone in lead V1 or V2. The R wave duration index is calculated by dividing the longer R wave duration in lead V1 or V2 by the QRS complex duration. A R/S amplitude index of less than 0.3 and R wave duration index of less than 0.5 may strongly suggest a PVC origin on the right side (Figure 5A).[16] The V2S/V3R amplitude ratio is calculated by dividing the amplitude of the S wave in lead V2 by that of R wave in lead V3. A V2S/V3R amplitude ratio of ≤1.5 can predict LVOT PVC origins and that of >1.5 RVOT PVC origins (Figure 5B).[26] This ECG algorithm is useful even when the precordial transition is in lead V3, and has been proven to be the most accurate among the previous ECG algorithms to differentiate RVOT PVC origins from LVOT PVC origins.

3.2 | RVOT and pulmonary artery origins

When PVCs originate from the RVOT, there are several ECG characteristics that can localize PVC origins in the RVOT.[28-30] First, a dominant R wave in lead I suggests a PVC origin in the posterior wall while a QS pattern suggests it is in the anterior wall of the RVOT.[30] Second, the presence of "notching" in the middle of the QRS complex strongly suggests a PVC origin in the RVOT free wall.[28,29] An RR′ (R-R′ >20 msec) pattern (the presence of "notching" in the middle of the QRS complex) in lead I and the inferior leads suggests that PVCs more likely originate from the free wall than the septum of the RVOT. Phased excitation from the RV free wall to the LV during PVCs originating from the RVOT free wall may account for this finding.[29] Third, the R wave amplitude in the inferior leads is likely to be lower, and the QRS duration in the inferior leads is likely to be longer during PVCs originating from the free wall than the septum of the RVOT.[29] These differences also may reflect phased excitation from the RV free wall to the LV during PVCs originating from the RVOT free wall, which can prolong the total QRS duration and decrease the absolute R wave magnitude in the course of activation of both ventricles. On the other hand, during PVCs originating from the RVOT septum, the two ventricles are excited nearly simultaneously, which would result in a shorter QRS complex duration and a higher absolute R wave magnitude. Fourth, a precordial transition at or earlier than lead V3 is unlikely in PVCs originating from the RVOT free wall.[28] Fifth, S waves in leads V1 to V3 are likely to be deeper during PVCs originating from the free wall than the septum of the RVOT (Figure 6).[29] The RVOT free wall is located closest to the electrodes of leads V1 to V3, and during PVCs originating from the RVOT free wall, the total activation vector from the PVC origins should go away from these electrodes. On the other hand, during PVCs originating from the RVOT septum, the main activation vector from the PVC origins should go away from the electrodes of leads V1 to V3, but a part of the activation vector should go toward those electrodes, resulting in smaller S wave amplitudes in leads V1 to V3 than during PVCs originating from the RV free wall.

TABLE 1 Electrocardiographic (ECG) characteristics of idiopathic premature ventricular contractions (PVCs)

Location of PVC origins	BBB	Axis	Precordial transition	Lead I	Lead V1	Lead V6	Specific ECG characteristics
Left ventricle							
ASOV							
LSV	LBBB>>RBB-B	RI>>LI	≤V2	rS, Rs	rS, RS	R	III/II ratio >0.9
RSV	LBBB	LI	≤V3	R	rS, RS	R	RBBB and RIA are rare. III/II ratio ≤0.9
L-RCC	LBBB	LI	≤V2-V3	R, rs	qrS	R	RBBB and RIA are rare. A qrS pattern in V1-V3 A QS with a notch on the downward deflection in V1
NSV	LBBB	LI	≤V3	R	rS, QS	R	Negative QRS in III (50%), Positive QRS in aVL
LVOT							
AMC	RBBB	RI>>LI	Early	R, Rs	qR, QR, Qr, qr, QS	R,Rs	LBBB is rare. Positive precordial concordance
Septum	LBBB=RBBB	LS=LI	≤V1-V2	R	Q, qR, Qr	R	Negative QRS in III (100%)
MA							
Ant to Lat	RBBB	RI	Early	rS	R	Rs,R	Late notching of R wave in the inferior leads Wide QRS, Positive precordial concordance
PS to Post	RBBB	LS	Early	R, Rs	R	Rs, R	Late notching of Q wave in the inferior leads (+) or (−) = post or PS Positive precordial concordance
Epicardial							
LV summit	RBBB>>LBB-B	RI	≤V2-V3	rS, rs, QS	R, QS, rS	R, Rs	MDI >0.55 (30%-40%)
Crux	LBBB>>RBB-B	LS	≤V1-V2	R	QS, Qr, R	Vari-able	QRS polarity reversal between V1 & V2, Positive concordance in V2-V6 MDI >0.55, slurred intrinsicoid deflection
Papillary muscles							
Posteromedial	RBBB	LI=RI	<V1	Rs, rS	qR	RS	qR or R waves in lead V1
Anterolateral	RBBB	RI	<V1	rS	qR	RS	R/S ratio ≤1 in lead V6
Fascicular							
Posterior	RBBB	LS=RS	<V1	Rs, rS, R	rsR′	RS	rsR′ waves in lead V1, QRS duration <130 ms
Anterior	RBBB	RI	<V1	rS	qR	Rs	QRS duration <130 ms
Upper septal	LBBB	Normal or right	V3	Rs	rS	Rs	Narrow QRS complex with VA dissociation
Right ventricle							
RVOT							
Septum	LBBB	LI>>RI	≤V3	rS, QS	rS	R	No notching in the limb leads
Free wall	LBBB	LI	≥V3	R	rS	R	Notching in the limb leads, Deep S waves in V1-V3
PA	LBBB	RI>>LI	>V3	rS, QS	rS, QS	R	aVL/aVR ratio >1
TA							
Septum	LBBB	LI, LS	<V3	R/r	QS	R	Positive, isoelectric, or multiphasic in aVL
Free wall	LBBB	LI, LS	>V3-4	R/r	rS	R	A notch in the middle of the QRS
ParaHisian	LBBB	LI, LS	<V3	R/r	QS	R	Positive, isoelectric, or multiphasic in aVL
Papillary muscles							
Posterior	LBBB	LS>>LI	≥V4-5	R/r	QS, rS	r	A notch in the precordial leads
Anterior	LBBB	LS>>LI	V4-5	R/r	rS, QS	r	A notch in the precordial leads
Septal	LBBB	LI	≤V2-V3	R/r	rS, QS	R	A notch in the precordial leads
Muscle bands							
Moderator band	LBBB	LS	>V4	r	QS, rS	r, rs, qs	Intrinsicoid deflection in the precordial leads <100 ms
Parietal band	LBBB	LI>>>LS	≤V3	R/r	QS, rS	R	Slow onset of the QRS complex, notch in the middle of the QRS
Septal band	LBBB	LI>>>RI	Variable	R/r or s (+)	QS, rS	R	Slow onset of the QRS complex

Abbreviations: AMC, aortomitral continuity; Ant, anterior; ASOV, aortic sinus of Valsalva; BBB, bundle branch block; Lat, lateral; LBBB, left bundle branch block; LI, left inferior; L-RCC, the junction between the left and right coronary cusps; LS, left superior; LSV, left sinus of Valsalva; LV, left ventricular; LVOT, left ventricular outflow tract; MA, mitral annulus; MDI, maximal deflection index; NSV, noncoronary sinus of Valsalva; PA, pulmonary artery; Post, posterior; PS, posteroseptal; RBBB, right bundle branch block; RI(A), right inferior (axis); RS, right superior; RSV, right sinus of Valsalva; RVOT, right ventricular outflow tract; TA, tricuspid annulus; VA, ventriculoatrial.

It has been reported that idiopathic PVCs can originate from a myocardial sleeve extending from the RVOT onto the pulmonary artery.[27,31,32] Because of the anatomical background, there are no specific ECG characteristics that can differentiate PVCs originating from the RVOT and pulmonary artery.

3.3 | Aortic root and AMC origins

Anatomically, the superior end of the LV myocardium (LV ostium) comes in direct contact with the aorta at the base of the aortic coronary cusps, and never extends above the aortic coronary cusps.[19] The aortic root including the ASVs and the junction between the left and right coronary cusps (L-RCC) and AMC constitute a part of the LV ostium,[19] and are located next to each other (Figure 4). The AMC literally indicates the place between the aorta and mitral annulus (MA). However, its definition may differ between the pathology and electrophysiology. For the pathology, the AMC indicates the left fibrous trigone where ventricular arrhythmias cannot arise. On the other hand, for the electrophysiology, the AMC indicates the endocardial LVOT in front of the junction between the aorta and MA where ventricular arrhythmias can arise. The ASVs are superior to the AMC and likely to be epicardial. The AMC is endocardial underneath the aortic valve and is located near the left anterior fascicle. Therefore, in general, ASV PVCs exhibit taller R waves in the inferior leads and a wider QRS than AMC PVCs (Figure 4). The presence of S waves in lead V6 may suggest a PVC origin in the AMC[17] probably because the S wave in lead V6 is consistent with an RBBB pattern, usually present in PVCs with an LV endocardial origin.[33] AMC PVCs usually exhibit an RBBB pattern, but rarely an LBBB pattern with an early precordial transition.[23] AMC PVCs typically exhibit an rS pattern in lead I and qR pattern in lead V1 (Figures 4 and 7),[34] and often exhibit a "rebound transition pattern" that is defined as a negative QRS polarity in lead V2, but with a positive QRS polarity in both leads V1 and V3. The "rebound transition pattern" has also been referred to as an "R wave pattern break in lead V2," which may suggest a PVC origin close to the anterior interventricular sulcus (anatomically opposite to lead V2) and adjacent to the proximal coronary arteries.[35] An MDI of more than 0.55 suggests the unlikelihood of PVC origins in the AMC.[36]

Although the three ASVs are located next to each other, idiopathic PVCs originating from each ASV may be differentiated by ECGs (Figure 4).[18,19] Anatomically, the left sinus of Valsalva (LSV) is the most superior structure and the noncoronary sinus of Valsalva (NSV) is the most inferior one among the three ASVs.[19] The LSV is located on the left of the right sinus of Valsalva (RSV) and NSV. Therefore, in general, when the location of PVC origin shifts from the NSV to the RSV and then LSV, the PVCs exhibit a more negative QRS polarity in lead I, increase in the R wave amplitude in the inferior leads, and counter-clockwise rotation of the precordial transition. Idiopathic PVCs originating from the RSV and at the L-RCC rarely exhibit an RBBB pattern, and idiopathic PVCs originating from the NSV always exhibit an LBBB pattern.[18,19,37-39] The R wave amplitude ratio in leads III to II (III/II R wave ratio) is useful for differentiating LSV PVCs from RSV PVCs.[18] When the III/II R wave ratio is > 0.9, PVCs are more likely to originate from the LSV. A "qrS" pattern in the right precordial leads and a QS morphology with notching on the downward deflection in lead V1 may be highly specific for an L-RCC PVC origin (Figure 4).[37,39] Both ECG features may basically reflect the same electrical phenomenon because the notch on the downward deflection of the QS morphology could become an "r" wave in the "qrS" pattern if it is tall enough to exceed the isoelectrical line.

The ECG characteristics of NSV PVCs are similar to those of RSV PVCs (Figure 4).[18,19,38] However, an S wave in lead III is often present during NSV PVCs although it is not during RSV PVCs. When the III/II R wave ratio is < 0.65, PVCs are more likely to originate from the NSV.

3.4 | LV summit and intramural LVOT origins

It should be challenging to define the ECG characteristics of PVCs originating from the LV summit and intramural LVOT for several reasons (Figure 7).[20,40,41] First, those regions are located close to the aortic root (Figure 4). Second, it is challenging to identify the exact sites of PVC origins in those regions by the current mapping technique and technologies because of the anatomical barriers such as an intramural region, close proximity of the left coronary arteries, and thick epicardial fat pads.[20,42] Third, a successful ablation is not a gold standard to identify the exact site of the PVC origins in these regions. Epicardial catheter ablation can eliminate intramural PVC origins by creating a deep lesion. Endocardial catheter ablation can eliminate intramural PVC origins and even epicardial PVC origins by creating a deep lesion (anatomical approach).[40,41,43] A successful combined ablation from endocardial and epicardial sites may suggest an intramural origin. For these reasons, most of the previous articles on the ECG characteristics in these regions have described the ECG characteristics that can predict a successful catheter ablation through endocardial and epicardial approaches.

Currently, only one article has described the differences in the ECG characteristics among PVCs originating from the LV summit, and intramural and endocardial LVOT.[41] According to that article, the QRS duration of LV summit PVCs is generally greater than that of endocardial and intramural LVOT PVCs. LV summit PVCs always exhibit a right inferior axis QRS morphology, whereas the endocardial and intramural LVOT PVCs sometimes exhibit a left inferior axis QRS morphology. In lead I, an rS pattern is dominant and the absence of Q waves is very rare during PVCs originating from the endocardial and intramural LVOT while an rS pattern and the presence of Q waves are half and half during LV summit PVCs. The III/II R wave and aVL/aVR S wave amplitude ratios and MDI are significantly greater in LV summit PVCs than endocardial LVOT PVCs. However, there are no significant differences in the III/II R wave and aVL/aVR S wave amplitude ratios or MDI between the intramural LVOT PVCs and endocardial LVOT or LV summit PVCs, suggesting that the ECG characteristics of the intramural LVOT PVCs might be in the middle between those of the endocardial LVOT PVCs and LV summit PVCs.

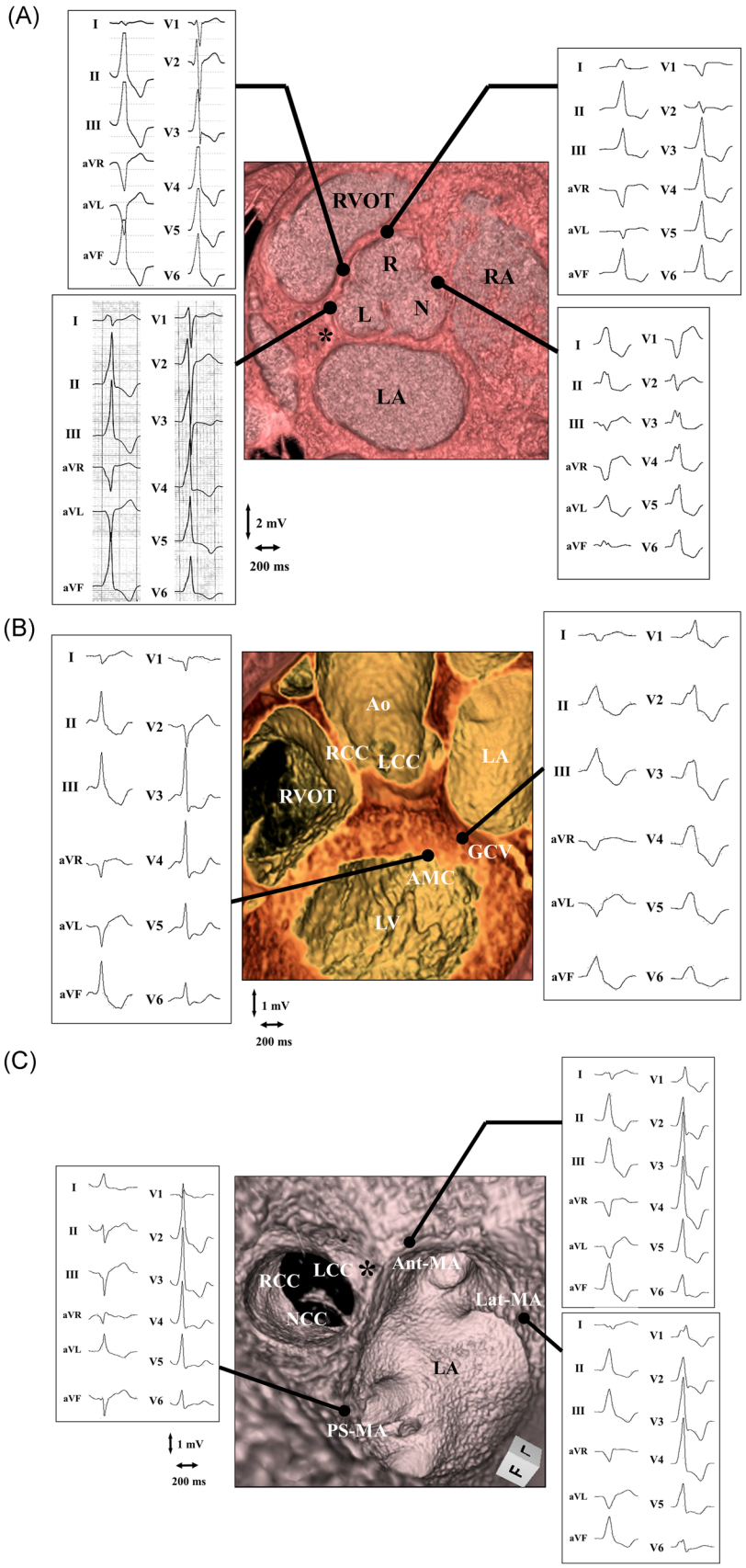


FIGURE 4 Computed tomography images and representative 12-lead electrocardiograms of the ventricular arrhythmias arising from the LV ostium. The asterisk indicates the left fibrous trigone. AMC, aortomitral continuity; Ant, anterior aspect; Ao, aorta; GCV, great cardiac vein; Lat, lateral aspect; L or LCC, left coronary cusp; LA, left atrium; LV, left ventricle; MA, mitral annulus; N or NCC, noncoronary cusp; PS, posteroseptal aspect; R or RCC, right coronary cusp; RA, right atrium; RVOT, RV outflow tract. This figure was cited from reference 19 with permission

TABLE 2 Major electrocardiographic algorithms predicting ventricular arrhythmia origins in the right or left ventricular outflow tract

	Authors	Year	Name	No. of Pts	Measures	% of V3 TZ Pts	Analysis of V3 TZ	Comparison with the other algorithms
1	Kamakura et al[15]	1998	V3-lead R/S ratio	40	PVCs only	N/A	–	–
2	Ouyang et al[16]	2002	R/S-wave amplitude index R-wave duration index	15	PVCs only	N/A	–	–
3	Betensky et al[21]	2011	V(2) transition ratio	40+21	PVCs & SR	100	+	–
4	Yoshida et al[22]	2011	TZ index	112	PVCs & SR	N/A	–	4>2
5	Cheng et al[24]	2013	R wave deflection interval & R wave amplitude index	82	PVCs only	38	+	–
6	Yoshida et al[26]	2014	V2S/V3R index	207	PVCs only	37	+	6>4>3=2

Abbreviations: N/A, not applicable; Pts, patients; PVCs, premature ventricular contractions; SR, sinus rhythm; TZ, transition zone.

Anatomically, the great cardiac vein (GCV) bisects the LV summit into a superior portion that is in close proximity to the proximal left coronary arteries and overlying epicardial fat (the inaccessible area) and an inferior portion that may be accessible to epicardial catheter ablation (the accessible area).[20] Because catheter ablation of PVCs originating from the inaccessible area is challenging and that of PVCs originating from the accessible area and the site below the GCV is highly successful, it would be important to know the differences in the ECG characteristics between PVCs originating from those areas. There are different ECG characteristics of the LV summit PVCs among the three areas of the LV summit that are reasonably understood in consideration of the anatomical relationships among the three areas.[20,42] First, the maximal amplitude of the R wave in the inferior leads is the highest in the inaccessible area PVCs, second in the GCV PVCs, and lowest in the accessible area PVCs. Second, an RBBB pattern always occurs in the accessible area PVCs and is dominant in the GCV PVCs but rare in the inaccessible area PVCs. Third, a very early precordial transition of the PVCs (earlier than in lead V1) is dominant in the accessible area PVCs and GCV PVCs, whereas it never occurs in the inaccessible area PVCs. Fourth, the QRS duration is significantly longer in the accessible area PVCs and GCV PVCs than in the inaccessible area PVCs. Fifth, the III/II R wave and aVL/aVR S wave amplitude ratios are significantly higher in the accessible area PVCs than in the GCV PVCs and inaccessible area PVCs. When PVCs are considered to originate from the LV summit, an RBBB pattern, transition zone earlier than lead V1, aVL/aVR ratio >1.1, and presence of S waves in lead V6 suggest with a high accuracy that PVCs should originate from a GCV site and the accessible area.[20,42]

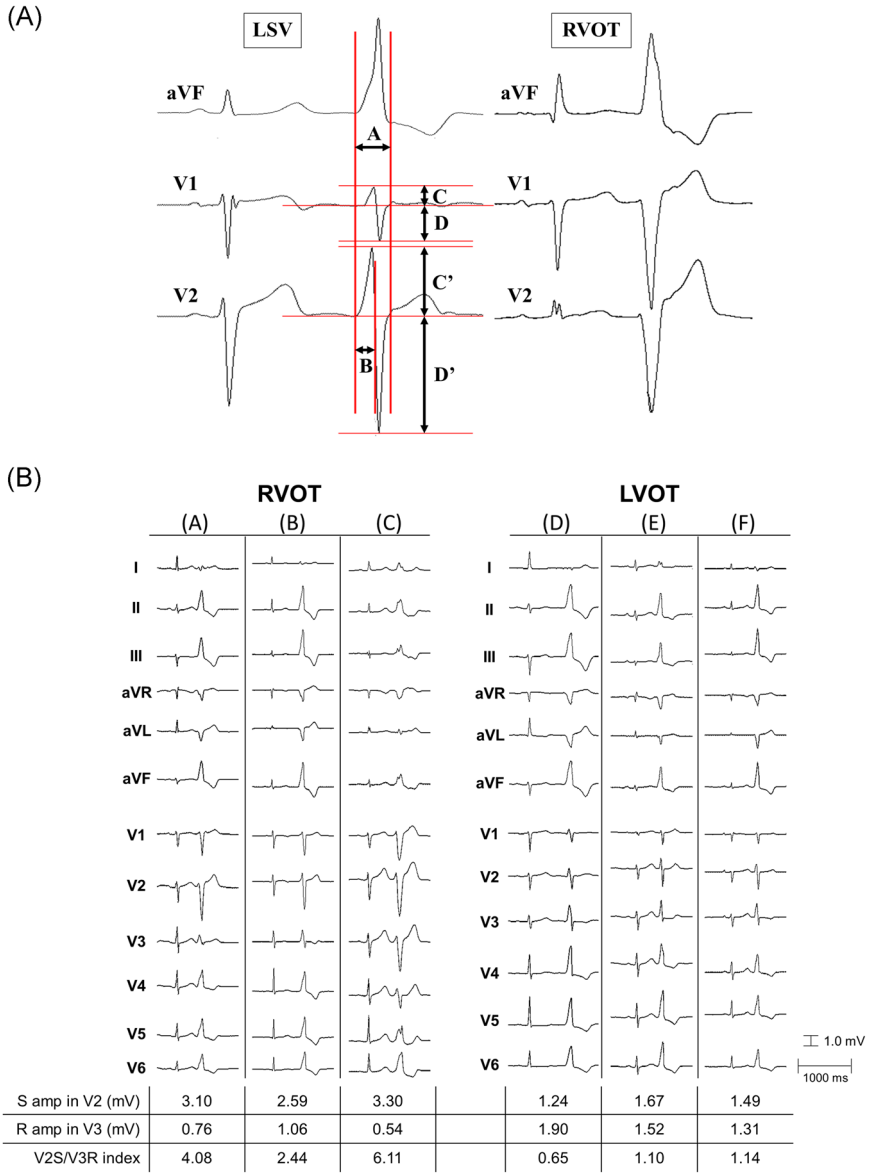
3.5 | Para-Hisian origins

There are multiple major sites of idiopathic PVC origins in the para-Hisian region such as the His bundle region in the RV, RSV, NSV, and LV septum below the membranous septum.[7,44-46] Anatomically, all of those sites are located close to each other, and the ECG characteristics of idiopathic PVCs originating from those sites are very similar. Idiopathic para-Hisian PVCs commonly exhibit a tall R wave in lead I, and III/II R wave ratio of less than 1. They typically exhibit an absence of QRS notching and a QS pattern in lead V1, but PVCs originating from the LV septum below the membranous septum sometimes exhibit an RBBB pattern.[46] In idiopathic para-Hisian PVCs, the QRS polarity in lead aVL is often positive. Even when it is negative, an S wave amplitude in lead aVL is smaller than that in lead aVR.

The prediction of the exact site of para-Hisian PVC origins by ECGs is not so accurate. However, it would be helpful for mapping and ablation of the para-Hisian PVCs to know the potential differences in the ECG characteristics among the sites in the para-Hisian region. The precordial transition should be later on the right side than on the left side. A negative QRS polarity in lead III never occurs during RSV PVCs while it often occurs during PVCs originating from the NSV, LV septum below the membranous septum, and right para-Hisian region. When the QRS polarity is positive in lead II but negative in lead III, it is called an inferior lead discordance.[47]

3.6 | Mitral annular origins

All PVCs originating from the MA exhibit an RBBB pattern and monophasic R or Rs in leads V2 to V6 (Figure 8).[8,48] Because the origins of all MA PVCs are located in the posterior portion of the LV, distant from the precordial electrodes, the activation from the MA PVC origins propagates toward these electrodes, resulting in an early precordial transition and concordant positive QRS pattern in leads V2 to V4 during MA PVCs. The ECG characteristics are very helpful for predicting sites of MA PVC origins.[8,48] The polarity of the QRS complex in the inferior and lateral leads (I and aVL) is positive and negative in anterolateral MA PVCs while it is negative and positive in posterior and posteroseptal MA PVCs, respectively. MA PVCs originating from the free wall of the MA are characterized by a longer QRS duration sometimes with pseudo-delta waves and notching in the late phase of the R or Q wave in the inferior leads, which may result from phased excitation from the LV free wall to the RV (Figure 8). These ECG characteristics are not observed in the posteroseptal, anterior, and anteromedial MA PVCs. Posterior MA PVCs exhibit a dominant R wave in lead V1, resulting in a precordial transition of <lead V1 whereas posteroseptal MA PVCs exhibit a negative QRS component in lead V1 (qR, qr, rs, or rS). This is because



	RVOT (A)	RVOT (B)	RVOT (C)	LVOT (D)	LVOT (E)	LVOT (F)
S amp in V2 (mV)	3.10	2.59	3.30	1.24	1.67	1.49
R amp in V3 (mV)	0.76	1.06	0.54	1.90	1.52	1.31
V2S/V3R index	4.08	2.44	6.11	0.65	1.10	1.14

FIGURE 5 A, Examples of an electrocardiographic analysis of ventricular arrhythmias (VAs). The first beats are sinus and the second beats are ventricular arrhythmias originating from the left sinus of Valsalva (LSV) and right ventricular outflow tract (RVOT). (A) indicates the total QRS duration, (B) the longer R-wave duration in lead V1 or V2, determined in lead V2 from the QRS onset to the R-wave intersection point where the R-wave crosses the isoelectric line, (C) the R-wave amplitude, measured from the peak to the isoelectric line, and (D) the S-wave amplitude measured from the QRS nadir to the isoelectric line. The R/S wave amplitude ratio in lead V2 (C'/D') is greater than that in lead V1 (C/D) and C'/D' is determined as the R/S wave amplitude index. The R/S amplitude index is less than 0.3 and the R wave duration index less than 0.5 during RVOT VAs, whereas they are not during LSV VAs. This figure was cited from reference 25 with permission. B, The representative 12-lead electrocardiograms of the VAs originating from the ventricular outflow tract. The first beat is a sinus beat and the second is a premature ventricular contraction (PVC) in each panel (A-F). The S-wave amplitude in lead V2, R-wave amplitude in lead V3, and V2S/V3R index are listed below each panel. All RVOT PVCs exhibited a V2S/V3R index of >1.5, while all left ventricular outflow tract (LVOT) PVCs exhibited a V2S/V3R index of ≤1.5. The PVCs were successfully ablated in the RVOT septum (A,B), RVOT free wall (C), left coronary cusp (D), right coronary cusp (E), and aortomitral continuity (F). This figure was cited from reference 26 with permission

the activation from the posteroseptal MA PVC origin propagates toward the left and away from the electrode of lead V1 which is positioned horizontally to the ventricular septum. The Q or S wave amplitude ratio of leads III to II is greater for posteroseptal MA PVCs than posterior MA PVCs because the activation from the posteroseptal MA PVC origins propagates more toward the left than that from the posterior MA PVC origins.

3.7 | Tricuspid annular origins

All PVCs originating from the tricuspid annulus (TA) exhibit an LBBB QRS morphology and positive QRS polarity in leads I, V5, and V6 (Figure 9).[7] Because the TA PVC origins are located on the right anterior side of the heart, and the activation propagating from TA PVC origins toward the apex generates a positive QRS polarity in

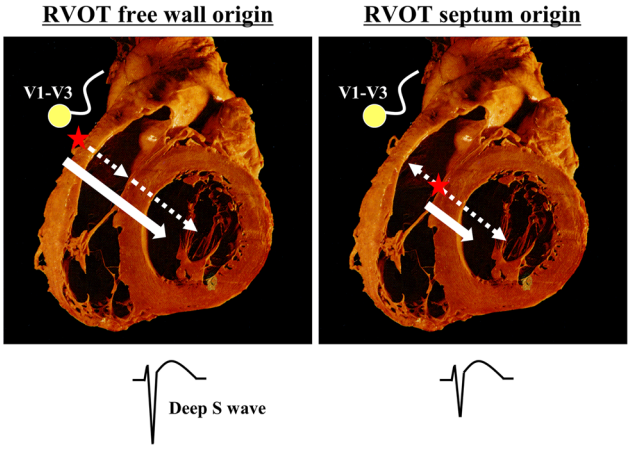


FIGURE 6 Possible mechanism responsible for the higher-amplitude S waves in leads V1 to V3 in patients with ventricular arrhythmias originating from the RVOT free wall than septum. This figure was modified and cited from reference 27 with permission (courtesy from the UCLA collection). RVOT, right ventricular outflow tract

leads V5 and V6. The R wave in lead I is usually taller during TA PVCs than during RVOT PVCs because the TA is located more rightward and inferior to the RVOT. For the same reason, a positive QRS polarity in all of the inferior leads is rare in TA PVCs but common in all RVOT PVCs. During TA PVCs, a QS or rS pattern in lead aVL is rare, and the QRS polarity in lead aVL is positive in almost all TA PVCs, which is not the case for RVOT PVCs.

Among all TA PVCs, the QRS duration and Q or S wave amplitude in each of the leads V1 to V3 are greater in TA PVCs originating from the free wall of the TA than in those from the septal wall of the TA.[7] Septal TA PVCs exhibit an early precordial transition (lead V3), narrower QRS duration, and QS in lead V1 with the absence of notching in the inferior leads while the free wall TA PVCs are associated with a late precordial transition (>lead V3), wider QRS duration, the absence of Q waves in lead V1, and the presence of

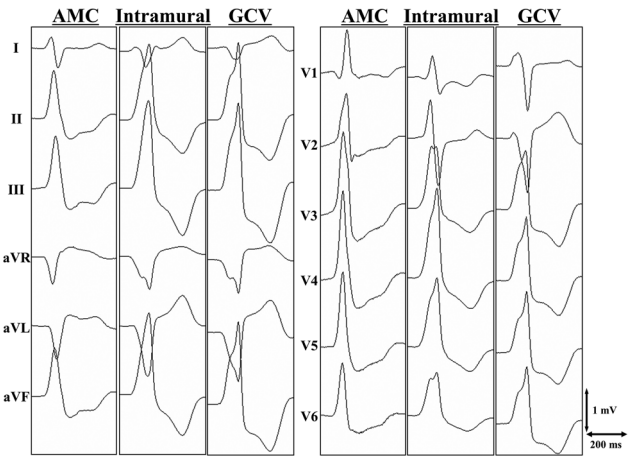


FIGURE 7 The representative 12-lead electrocardiograms of the ventricular arrhythmias originating from the aortomitral continuity (AMC), intramural site, and great cardiac vein (GCV). This figure was cited from reference 41 with permission

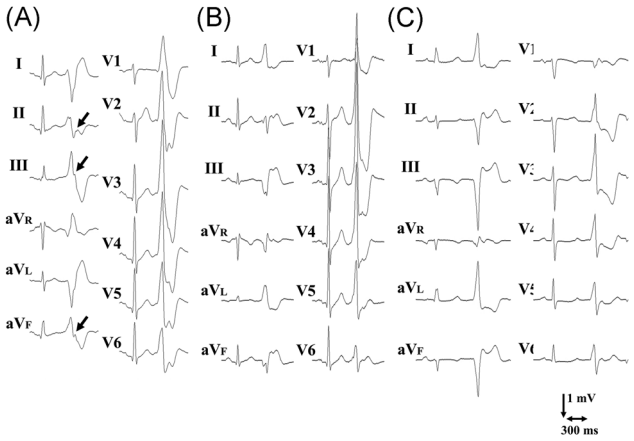


FIGURE 8 The representative 12-lead electrocardiograms of the premature ventricular contractions originating from the anterolateral (A), posterior (B), and posteroseptal (C) aspects of the mitral annulus. The arrows indicate "notching" of the late phase of the QRS complex in the inferior leads. This figure was cited from reference 13 with permission

notching in the inferior leads (the timing of the second peak of the notched QRS complex in the inferior leads corresponds precisely with the LV free wall activation; Figure 9). A negative QRS polarity in the inferior leads predicts PVC origins in the posterior aspect of the TA, and otherwise, VA origins in the mid to anterior aspects of the TA are suggested.

3.8 | Papillary muscle and fascicular origins

Idiopathic PVCs originating from the posteromedial and anterolateral papillary muscles (PAMs) in the LV exhibit RBBB and right inferior and left or right superior axis QRS morphologies, respectively (Figure 2).[6,9,49-51] Idiopathic PVCs originating from the anterolateral PAMs sometimes exhibit an inferior lead discordance with a negative and positive QRS polarity in leads II and III, respectively.[47] Idiopathic PVCs originating from the posterior (inferior) or anterior RV PAMs more often exhibit a superior axis with a late precordial transition (>lead V4) as compared with septal (superior) RV PAM VAs, which more often exhibit an inferior axis with an earlier precordial transition (≤lead V4; Figure 10).[52] Idiopathic PVCs originating from the posterior RV PAMs sometimes exhibit an inferior lead discordance with a negative and positive QRS polarity in leads III and II, respectively.[47]

Because of the close anatomical relationship, it is important to distinguish PAM PVCs from MA PVCs and PVCs originating from the LV fascicles by ECGs (Figure 2).[6] An Rs or rS in lead I, qR in lead aVL, and Q in lead V1 are common ECG features that can accurately distinguish PAM and LV fascicular PVCs from MA PVCs irrespective of the QRS axis and regions of PVC origins.[6] An rS in lead aVL and absence of a positive precordial concordance pattern or R/S ratio ≤1 in lead V6 can do so for only inferior QRS axis PVCs originating from the LV anterolateral region or superior QRS axis PVCs originating from the posterior region, respectively.[6,53] However, the ECG features are

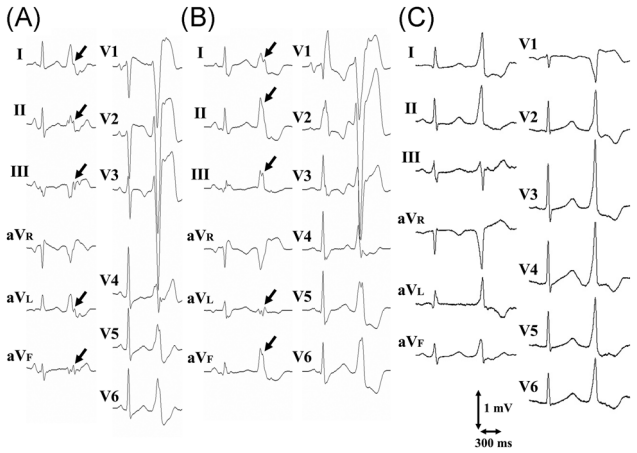


FIGURE 9 The representative 12-lead electrocardiograms of the premature ventricular contractions originating from the posterolateral (A), anterior (B), and anteroseptal (C) aspects of the tricuspid annulus. The arrows indicate "notching" of the late phase of the QRS complex in the limb leads. This figure was cited from reference 13 with permission

very similar for PAM and LV fascicular PVCs, and an R/S ratio ≤1 in lead V6 in the LV anterolateral region and a QRS duration >160 ms, and qR or R waves in lead V1 (as compared with an rsR' for fascicular PVCs) in the LV posteroseptal region may be the only reliable predictors for differentiating PAM PVCs from LV fascicular PVCs.[6,54] A QRS duration of less than 130 ms strongly suggests fascicular PVCs in both the LV anterolateral and posteroseptal regions.[53-55]

3.9 | Parietal and septal band origins

The ECG characteristics of idiopathic PVCs originating from the parietal band (PB) are similar to those of idiopathic PVCs originating

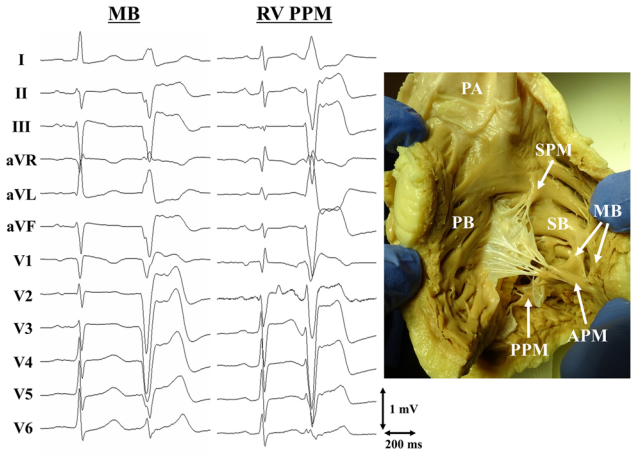


FIGURE 10 Twelve-lead electrocardiograms of the premature ventricular contractions originating from the moderator band (MB; left panel), and posterior papillary muscle in the right ventricle (middle panel), and autopsy heart exhibiting the muscular bands and papillary muscles in the right ventricle. APM, anterior papillary muscle; PA, pulmonary artery; PB, parietal band; PPM, posterior papillary muscle; SB, septal band; SPM, septal (superior) papillary muscle

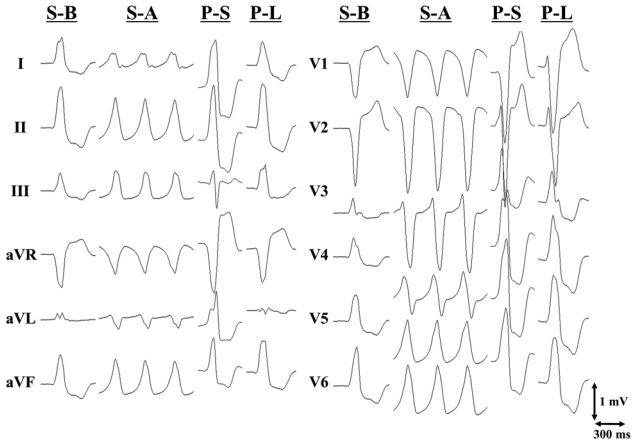


FIGURE 11 The representative 12-lead electrocardiograms of ventricular arrhythmias originating from the infundibular muscles. P-S (L) indicates the septal (lateral) portion of the parietal band; and S-A (B), the anterior (basal) portion of the septal band. This figure was cited from reference 57 with permission

from the free wall of the RVOT and the anterior aspect of the TA while those of idiopathic PVCs originating from the septal band (SB) are similar to those of idiopathic PVCs originating from the anteroseptal aspect of the TA (Figure 11).[56,57] The QRS morphologies of PB PVCs are characterized by an LBBB and typically left inferior and sometimes superior axis pattern while those of SB PVCs are characterized by an LBBB and typically left inferior and sometimes right inferior axis pattern. PB PVCs always exhibit R or r waves in lead I while s waves are sometimes present in lead I with SB PVCs. PB and SB PVCs always exhibit a III/II R wave ratio of less than 1, a QS pattern in lead aVR, R waves in lead V6, and presence of a notch in the middle of the QRS. PB and SB PVCs often exhibit an early precordial transition (≤lead V3). PB and SB PVCs sometimes exhibit a slow onset of the QRS complex, suggesting a PVC origin deep inside those muscle bands. There are no significant differences in any ECG characteristics between the PB and SB PVCs.

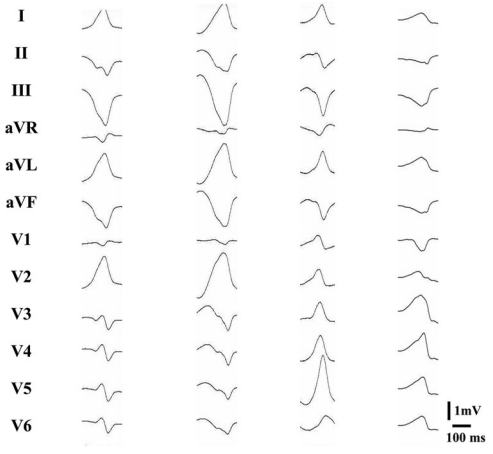


FIGURE 12 Twelve-lead electrocardiograms of ventricular arrhythmias originating from the crux of the heart. This figure was cited from reference 59 with permission

3.10 | Moderator band origins

Idiopathic ventricular fibrillation can be triggered by PVCs originating from the moderator band (MB) that exhibit a distinctive ECG morphology, LBBB and left superior axis QRS morphology, sharp downstroke of the QRS in the precordial leads, and relatively narrow QRS duration (Figure 10).[58] MB PVCs not only have a late precordial transition pattern, typically after lead V4, but also the transition is always later than that of the sinus QRS. Among the idiopathic RV PVCs, a late precordial transition and superiorly directed nature are helpful for distinguishing MB PVCs from PVCs originating from the RV base or septum.[58] Although the precordial transition is later during MB PVCs than PVCs originating from the septal RV PAM, there are no ECG features that clearly distinguish MB PVCs from anterior and posterior RV PAM PVCs likely because of the similar insertion on the RV free wall and highly variable anatomy (Figure 10). MB PVCs sometimes exhibit an inferior lead discordance with a negative and positive QRS polarity in leads III and II, respectively.[47] It is also noted that MB PVCs exhibit varied ECG morphologies because of the altitudinal orientation of the heart, and the variation in the origin and insertion of the MB as well.

3.11 | Cardiac crux origins

Idiopathic PVCs arising from the crux of the heart exhibit a left superior axis QRS morphology with deeply negative deltoid waves (QS pattern) in the inferior leads and an early precordial transition

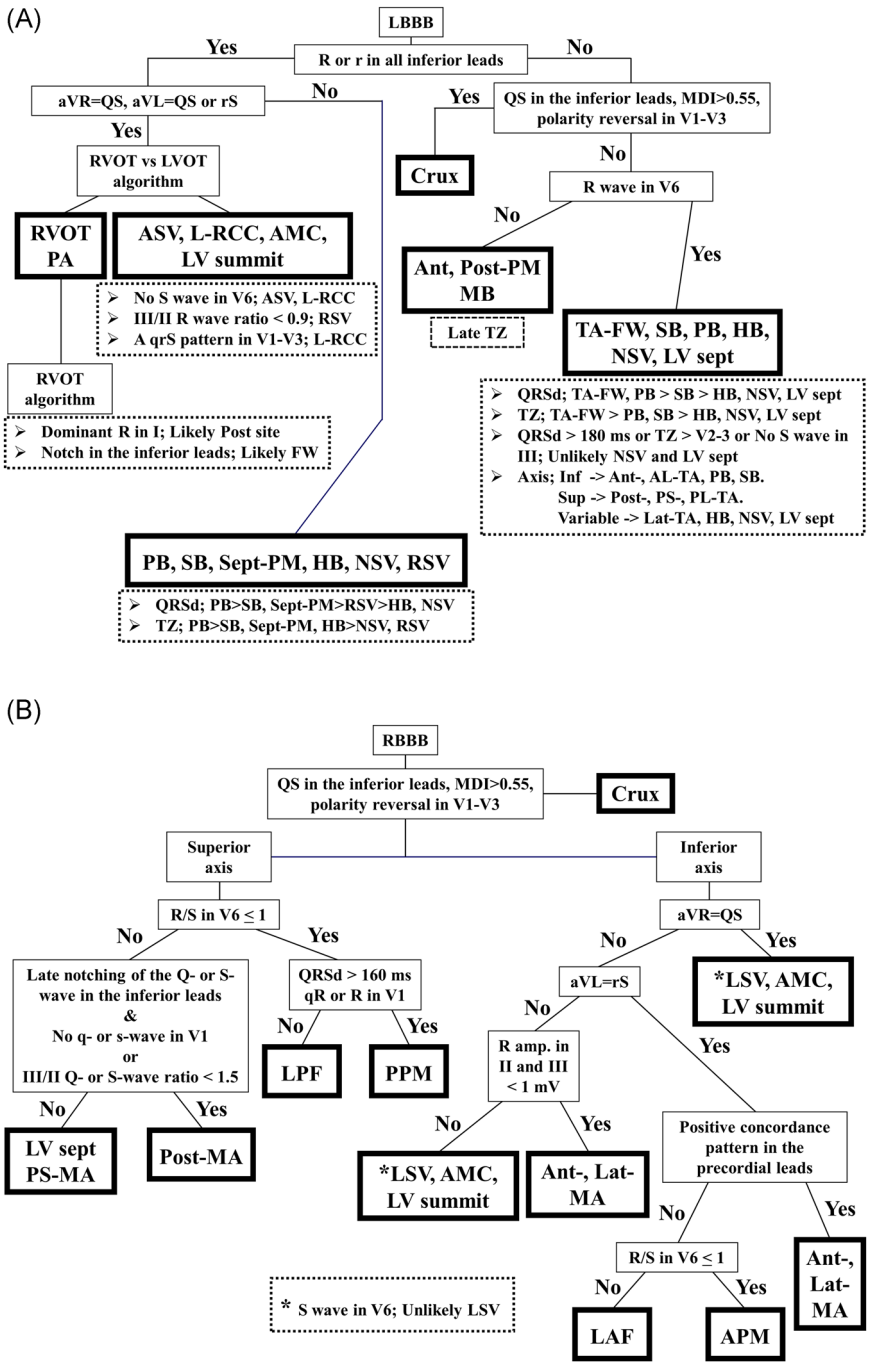


FIGURE 13 An electrocardiographic algorithm to localize sites of idiopathic ventricular arrhythmia origins. AL, anterolateral; AMC, aortomitral continuity; Ant, anterior; APM, anterolateral papillary muscle; ASV, aortic sinus of Valsalva; FW, free wall; HB, right ventricular His bundle region; Inf, inferior; Lat, lateral; LAF, left anterior fascicle; LBBB, left bundle branch block; LPF, left posterior fascicle; LSV, left sinus of Valsalva; L-RCC, the junction between the left and right coronary cusps; LV, left ventricular; LVOT, LV outflow tract; MA, mitral annulus; MB, moderator band; MDI, maximum deflection index; NSV, noncoronary sinus of Valsalva; PA, pulmonary artery; PB, parietal band; PL, posterolateral; PM, papillary muscle; Post, posterior; PPM, posteromedial papillary muscle; PS, posteroseptal; RBBB, right bundle branch block; RSV, right sinus of Valsalva; RVOT, right ventricular outflow tract; SB, septal band; sept, septum; Sup, superior; QRSd, QRS duration; TA, tricuspid annulus; TZ, precordial transition zone

(a prominent R wave in lead V2), which may be associated with a polarity reversal between leads V1 and V2 (Figure 12).[59] It is noted that a QS or large S wave is possible in lead V6 although crux PVCs arise from the LV base. These ECG characteristics are similar to those associated with maximally pre-excited posteroseptal accessory pathways because the latter insert into the same area of the LV as the origins of crux PVCs. This eccentric ECG pattern may be explained by the following mechanism. The activation from the crux epicardially propagates to the ventricular apex first where it enters the Purkinje network in the endocardium. Thereafter, it propagates rapidly throughout the ventricle, resulting in a major negative deflection of the QRS in lead V6. The MDI[11] is always greater than 0.55.[59]

4 | ECG ALGORITHMS

A lot of ECG algorithms for predicting the location of an idiopathic PVC origin have been proposed.[6-8,16-18,20-22,24,26,30,41,42,45,53-55,60] However, most of them were developed to differentiate sites of PVC origins in a specific region such as the ventricular outflow tract, right ventricular His bundle region, and so forth, and they can be less accurate or cannot be used in an ECG diagnosis of overall idiopathic PVCs. In addition, in recent years, several major anatomical sites of idiopathic PVC origins such as muscular bands have been newly recognized.[56-58] Therefore, a new ECG algorithm that can cover all major idiopathic PVC origins is needed in clinical practice. The author has proposed such an ECG algorithm in this article by summarizing the major specific ECG features and also taking several things into consideration (Figure 13). First, this ECG algorithm starts with a diagnosis of a bundle branch block pattern, which is the most common ECG feature to predict sites of PVC origins. Second, this ECG algorithm does not differentiate the detailed sites of PVC origins in some regions when it cannot be so accurate because of the close proximity and preferential conduction. Instead, several ECG features suggesting the likelihood of predicting the sites of PVC origins are noted. Third, in this ECG algorithm, the use of absolute numbers and detailed features in the precordial leads is avoided if possible because they are susceptible to the patient's physique.

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